DOI: 10.17707/AgricultForest.63.1.29

Mahmoud Reza KARIMISHAHRI, R.C. SHARMA[1]

INHERITANCE OF RESISTANCE TO MAYDIS LEAF BLIGHT IN MAIZE

SUMMARY

Maize suffers from the threat of various diseases resulting in considerable yield losses. One of them is maydis leaf blight (MLB) an economically important disease incited by *Bipolaris maydis* (Nisikado and Miyake) Shoemaker. Analysis of variance for combining ability indicated that both GCA and SCA variances were highly significant for disease reaction studied. High GCA variance for disease reaction suggested operation of additive gene action**.** Out of the 6 parents 3 exhibited –ve and 3 exhibited +ve GCA effects for resistance to *B. maydis*. Resistance in inbred V-17 had the highest negative GCA effects hence it is the best general combiner for resistance. The estimates of SCA effects indicated several crosses showing -ve SCA effects. Out of the 15 hybrids only 5 hybrids showed significantly –ve SCA effects. Highest estimates of SCA effects were observed in the cross V-335 x V-13 followed by V-327 x V-17 and V-128 x V-17. Parent V-13 (with significant negative GCA effect) when crossed with parent V-335 (with significant negative GCA effects) gave hybrid V-335 x V-13 which had significant negative SCA effects. Parent V-17 (with significant negative GCA effect) when crossed with parent V-128 (with significant positive GCA effects) resulted in a hybrid which had significant negative SCA effects. Amongst resistant x resistant cross only V-335 x V-13 indicated heterosis both over mid parents and better parental values. The crosses involving V-13, V-17 and V-335 with susceptible lines indicated maximum heterosis for resistance. While the mean effects were highly significant for the eight resistant crosses the a and d effects were significant for only 4 crosses of the a and d effects. Though each of the three digenic epistatic effects dd was playing a greater role followed by ad effects in the resistance crosses. In V-335 x V-13 cross, dd effect was significant and negative. The relatively high magnitude and positive effects of dd in most of the crosses indicated that these interactions was enhancing susceptibility, while considering the CM-128 x V-335, CM-128 x V-13, CM – 128 x V-128, CM-128 x V-17 and V-327 x V- 335 crosses were observed that ad and dd effects were highly significant and positive value.

Keywords: *India, GCA, SCA, Maize, Bipolaris maydis*

INTRODUCTION

Like other extensively cultivated crops, maize suffers from several diseases as well. In India 61 diseases have been reported on maize (Payak and

[1]Mahmoud Reza Karimishahri (corresponding author: mrkarimi@iripp.ir), R.C. Sharma, Division of Plant Pathology, Indian Agricultural Research Institute, New Delhi, INDIA

Sharma, 1985). Among them Maydis Leaf Blight (MLB) prevalent in many parts of India, is a major threat to maize cultivation when grown in warm and humid climates. Maize has great genetic diversity for resistance to pathogens, which makes the use of resistant cultivars the most economic and efficient form of disease control (Balmer & Pereira, 1987; Silva, 2001).

A logical requisite to launch a breeding programme for resistance to the disease is to investigate the mode of inheritance and to described the genetic variance present. Knowledge concerning the inheritance of characters showed increases the effectiveness of selection for this trait. Pate (1954) and Van Eijnatten (1961) have indicated that reaction to the disease shows a continuous variation in maize population and behaves as a quantitative trait. Although individual gene effects usually are not measurable in quantitative characters, statistical procedures have been developed. Since the development and use of resistant genotypes are the best means of obtaining practical control of foliar diseases, genetic information relating to host resistance would provide more relevant basis for making breeding decisions. The first recorded work on the inheritance of disease reactions to *H. maydis* is that of Ullstrup (1941) who reported that susceptibility is inherited as a monogenic recessive trait. Pate and Harvey (1954) observed a wide range of reactions which suggested polygenic inheritance.

Falugyi and Olorode (1984) demonstrated at resistance in two varieties is homogenic recessive. The genes for resistance in the two varieties are allelic. Thompson and Bergquist (1984) reported resistance in seedling stage was recessive but the mature plant resistance being independent of seedling reaction. The resistance could be explained by additive effects, is concerned with relatively few loci, and may be incorporated into susceptible inbreds by backcrossing. The application of concepts of heterosis, general and specific combining abilities (GCA and SCA, respectively) has been utilized for grain-producing crop breeding; GCA is relatively more important than SCA for nonselected endogamic lines, while the opposite is true for previously-selected lines (Sprague & Tatum, 1942; Hallauer & Miranda Filho, 1988; Nass et al., 2000). These concepts are useful both for the characterization of lines in crosses and for establishing heterotic standards between maize populations (Hallauer & Miranda Filho, 1988; Beck et al., 1990; Crossa et al., 1990; Han et al., 1991; Vasal et al., 1992), and in maize disease genetic resistance studies (Nelson & Scott, 1973; Lim & White, 1978; Callaway et al., 1990). Lingam *et al.* (1989) reported that analysis of data on disease severity revealed highly significant GCA and SCA variances, with the GCA component higher, indicating the predominance of additive gene action. Dey *et al*. (1989) revealed that the general combining ability (GCA) component is more important than the specific combining ability (SCA) component in the inheritance of resistance. The resistant lines generally had lower GCA effects and the crosses between moderately resistant and susceptible lines had negative SCA values.

MATERIAL AND METHODS

The present study was undertaken to find out the pathogenic reaction under field conditions.

Field experiments

The study was conducted on six inbred lines known for their reaction to *Bipolaris maydis* (race O). Detailed pedigree and disease reaction of these lines are provided in (Table 1). These lines and their crosses including (reciprocals mixed) were grown in a Randomized Block Design with four replications under recommended agronomic practices. All these material namely, 6 parental inbreds, 15 F_1 crosses, 15 sets of F_2 progenies, 15 BC_1 and 15 BC_2 progenies were planted in rows of 5 m length in replicated trials in disease nursery conducted during Kharif seasons at IARI field. Hundred plants for each of parents, F_1, F_2 and BC_1, BC_2 progenies per replication were sampled for disease scoring (a rating scale 1-5, Payak and Sharma, 1983).

Table 1: Source of seed material -*VPKAS (ICAR), Almora*

Parents	Pedigree
P_1	**CM 128**
P_2	**V 327** (P33C-ICH462-1/KC12/Suwan)-2/D1478)Ä-F- ##- Äb- Ä-1- Äb)
P_3	**V 335** (TZI-25-F-##- Äb- Ä-4-1- Äb- Äb)
P_4	**V 13** (P41-C13-MH-526-1-2-f-f-F-#- Ä-13-#-5- Äb)
P_5	**V 128** (VL 16 Ä-237-1-1-f- Ä-5-#- Äb)
P_6	**V 17** (Pob 45-C2-HC-151-7-1-1-2-1-2- ÄB-####- Ä-1-f-26-#-3- Äb Äb)

Mass multiplication of inoculum

Mass multiplication of inoculum was done as per procedure describing by Sharma (1983). Sorghum grains were soaked in fresh tap water for 24hrs, after through washing. Excess water from the soaked grains was drained off through several layers of cheesecloth. The grains were dispensed in flasks to one third of its value, following by autoclaving at 120^{o}C for 40 min. The sorghum grains were inoculated with freshly growing culture blocks of the pathogen, maintained at plant pathology laboratory DMR, IARI. Cultures were shaken every 2 days and incubated for 16-20 days at 24ºC and shaken every 2 days. Cultures were then mixed and air-dried at 20ºC inoculations were made by powder of sorghum grains into individual leaf whorls.

Disease scoring

The disease scoring was done by rating scale provided by Payak and Sharma (1983). The scale consists of five broad categories designated by numerals 1 to 5. The rating scale is as follows:

1.0- Very slight-to-slight infection, one or two to few scattered lesion on lower leaves.

2.0- Light infection, moderate number of lesion on lower leaves only.

3.0- Moderate infection, abundant lesions on lower leaves, few on middle leaves, extending to upper leaves.

4.0- Heavy infection, lesion abundant on lower and middle, extending to upper leaves.

5.0- Very heavy infection, lesions abundant on almost all leaves, plants prematurely dry or killed by the disease.

Transformation of data

The intensity of the disease shows a continuous variation and, therefore, it was measured on individual plant basis. A slightly modified version of the scale was suggested by Fisher and Yates (1963). Since the data were recorded on the rating scale of 1-5, it was subjected to transformation before analysis. Fisher and Yates (1963) transformation have been widely used in this kind of data. Nevertheless test of normality was also conducted to confirm the revision of the rating data to normality. The transformed values (for 9 classes) are as under:

Table 2: Actual and transformed value for 9 classes

Actual value	Transformed value
1.0	-1.49
1.5	-0.93
2.0	-0.57
2.5	-0.27
3.0	0.00
3.5	+0.27
4.0	+0.57
4.5	+0.93
5.0	+1.49

Analysis of variance

The analysis of variance (ANOVA) was carried out according to Randomized Complete Block Design (RCBD). The analyses were performed according to the procedure outlined by Hayman (1954a) and Griffing (1956a) using the mean values of each replication.

Combining ability analysis

The combining ability analysis was carried out according to Griffing's (1956b) Model-I (fixed effect model) and Method–II (parents and F_1s but no reciprocals). In this method experimental material is regarded as population, about which the inferences are to be drawn and combining ability effects of the parents could be compared when parents themselves are used as testers to identify good combiners. The combining ability effects (GCA or SCA) so obtained were tested for their significance, calculating 't' value as under and comparing it against the table 't' value (at 5% and 1% level of significance) at error degrees of freedom. The critical difference (CD) was calculated as a product of the standard error and 't' value.

Heterosis

The average of F_1 over replication were used for the estimation of heterosis expressed in percentage over mid parent values (MP), better parent values (BP) and standard check values. The computation of values was done as per Hayes et al. (1955)

Test of significance

Significance of heterosis was tested by 't' test. The calculated 't' was compared with table value of 't' at error degree of freedom from ANOVA comprising parents and F_1's at P=0. 05 and P= 0. 01. 't' value was estimated as follows (Fisher and Yates, 1963):

Estimate of gene action

All the six components of generation means, m, a, d, aa, ad and dd representing mean, additive, dominance, additive x additive, additive x dominance and dominance x dominance gene effects respectively were calculated from the population means of P_1, P_2, F_1 based on the method given by Hayman (1958).

RESULTS AND DISCUSSION

Disease reaction

The disease was recorded for parental, F_1, F_2, BC_1, BC_2 following 1-5 disease scoring method, where 1 indicates no disease and 5 indicates maximum disease. The observation were pooled and analyzed. For calculating the disease intensity individual plants were scored (Table 3).

Table 3: Mean performance of the parental, F1, F2 and backcross generation for *Bipolaris maydis* (original and transformed scale)

Disease rating (original scale)					**Disease rating (original scale)**					
	P1	P2	F1	F2	P1	P2	F1	F2	BC1	BC2
CM-128x V327	2.92	3.48	3.08	3.01	-0.04	0.27	0.04	0.01	-0.01	0.15
CM-128xV-335	2.92	2.25	2.11	2.30	-0.04	-0.42	-0.52	-0.40	-0.25	-0.21
CM-128 x V-13	2.92	2.74	2.32	2.58	-0.04	-0.15	-0.39	-0.23	-0.22	-0.37
CM-128xV-128	2.92	3.28	2.39	2.84	-0.04	0.16	-0.34	-0.09	-0.10	-0.17
CM-128xV17	2.92	2.10	1.87	2.53	-0.04	-0.53	-0.69	-0.27	-0.29	-0.60
V-327xV-335	3.48	2.25	2.54	2.89	0.27	-0.42	-0.25	-0.06	0.03	-0.05
V-327xV-13	3.48	2.74	2.53	2.73	0.27	-0.15	-0.26	-0.15	-0.08	-0.36
V-327xV-128	3.48	3.28	3.11	3.55	0.27	0.16	0.06	0.32	0.17	-0.19
V-327xV17	3.48	2.10	1.83	2.38	0.27	-0.53	-0.63	-0.36	-0.15	-0.70
V-33xV-13	2.25	2.74	1.28	2.23	-0.42	-0.15	-0.60	-0.45	-0.63	-0.40
V-335xV-128	2.25	3.28	2.04	2.42	-0.42	0.16	-0.57	-0.33	-0.29	-0.21
V-335xV17	2.25	2.10	1.41	1.83	-0.42	-0.53	-1.06	-0.71	-0.60	-0.66
V-1xV-128	2.74	3.28	2.05	2.32	-0.15	0.16	-0.56	-0.39	-0.37	-0.31
V-13xV17	2.74	2.10	1.51	1.92	-0.15	-0.53	-0.97	-0.65	-0.64	-068
V-128xV17	3.28	2.10	1.56	2.06	-0.15	-0.53	-0.92	-0.56	0.43	-0.65

Analysis of variance for the design of the experiment

The statistical analysis was carried out for disease reaction. Analysis of variance is given in (Table 4) for one character. The 'F' test indicated that variance due to parents and their progenies were significant for disease reaction at 1% probability. The analysis of variance revealed that the variance within parents and their progenies were highly significant for disease reaction studied.

Table 4: Analysis of variance for randomized block design

	Mean original scale		**Mean transformed scale**
SOURCE	D F	MSS	MSS
REPLICATION	3	1.10**	0.09**
TREATMENT	65	1.13**	0.37**
PARENTS	5	1.19**	0.40**
F_1	14	1.26**	0.44**
F_2	14	0.81**	0.29**
BC_1	14	1.06**	0.24**
BC_2	14	1.00**	0.34**
BETWEEN GROUPS	4	0.10	0.02
ERROR	195		
CD at 1%		0.562	0.249

**Significant at 1%

Combining ability effects

The disease data both on original and transformed scale were subjected to combining ability analysis following Griffing's(1956a). Method II, modal I was utilized in diallel cross analysis for combining ability analysis of the maydis leaf blight in maize.

Analysis of variance for combining ability

Analysis of variance for combining ability indicated the both GCA and SCA variances were highly significant for disease reaction studies. General combining ability variances was approximately five times higher in magnitude than the specific combining ability variance (Table 5). These data thus indicated that, although, variance due to GCA and SCA were important for disease resistant, major role in the expression of disease reaction in the present set of material is played by GCA. High GCA variance was found for disease reaction suggested the operation of additive gene action for this character**.**

Table 5: Analysis of variance for combining ability for reaction to *Bipolaris maydis*

SOURCE	DF	MSS
GCA	5	0. 997**
SCA	15	0. 195**
ERROR	0.195	0.024

**Significant at 1%

General Combining Ability effects

The estimates of GCA effects of the six parents in respect of disease reaction are given in (Table 6). Out of the 6 parents included in the present study 3 exhibited –ve and 3 exhibited +ve GCA effects for resistance to *B. maydis*. Resistance in inbred V-17 has the highest negative GCA effects of all the parents hence it is the best general combiner for resistance resulting in low infection. V-335 and V-13 also showed significant negative GCA effects. Inbreds CM-128, V-327 and V-128 showed significant positive GCA effects and are poor general combiners for resistance. V-128 and V-327 have significant positive GCA effects, and thus transmitted susceptibility to their progenies. Similar results were obtained on both original and transformed scale.

Table 6: Estimates of general combining ability effects of the parental lines for *Bipolaris maydis*

Inbred lines	Orginal scale	Transformed scale
CM-128	0.184**	0.101*
V-327	0.488**	0.287**
V-335	-0,281**	-0.132**
V-13	-0.121*	-0.035ns
V-128	0.198**	0.098*
V-17	-0.468**	-0.318
CD	At 5%	at 1%
SEgca	0.098	0.128

Specific Combining Ability effects

The estimate of SCA effects are presented in (Table 7). It was observed that several crosses showed -ve SCA effects. Out of the 15 hybrids only 5 hybrids showed significantly –ve SCA effects. Highest estimates of SCA effects were observed in the cross V-335 x V-13 (resistant x resistant) followed by V-327 x V-17 and V-128 x V-17 (susceptible x resistant). The V-17 as a good general combiner for resistance has transmitted in resistance with susceptible combination, while the cross V-335 x V-13 (resistant x resistant) produced the resistant hybrid as expected. Parent V-17 (with significant negative GCA effect) when crossed with parent V-327 (with significant positive effect) gave hybrid V-327 X V-17 which had significant negative SCA effects. Parent V-13 (with significant negative GCA effect) when crossed with parent V-335 (with significant negative GCA effects) gave hybrid V-335 x V-13 which had significant negative SCA effects. Parent V-17 (with significant GCA effect) when crossed with parent V-128 (with significant positive GCA effects) resulted in a hybrid which had significant negative SCA effects. Results based on transformed scale were similar to original scale. All three above crosses showed the negative SCA effect.

Table7: Estimate of specific combining ability effects of the 15 crosses for *Bipolaris maydis* at field condition

	Sca effects		**Mean Rating**		
Crosses	Original scale	Transformed scale	Original scale	Transformed scale	Reaction Type
CM-128xV-327	0.102ns	0.053ns	3.08	0.04	Susceptible
CM-12xV-335	-0.095ns	-0.087ns	2.11	-0.52	Intermediate
CM-128xV-13	-0.049ns	-0.057ns	2.32	-0.39	Intermediate
CM-128xV-128	-0.300*	-0.142*	2.39	-0.34	Intermediate
CM-128xV17	-0.151ns	0.072*	1.87	-0.69	Resistance
V-327xV-335	0.025ns	0.010ns	2.54	-0.25	Susceptible
V-327 xV-13	-0.140ns	0.113ns	2.53	-0.26	Susceptible
V-327xV-128	0.118ns	0.078ns	3.11	0.06	Susceptible
V-327xV17	-0.492**	0.202**	1.83	-0.63	Resistance
V-335xV-13	-0.622**	-0.037**	1.28	-0.60	Resistance
V-335xV-128	-0.181ns	-0.137*	2.04	-0.57	Intermediate
V-335xV17	-0.146ns	-0.210**	1.41	-1.06	Resistance
V-13xV-128	-0.329*	-0.221**	2.05	-0.56	Intermediate
V-13xV17	-0.206ns	-0.217**	1.51	-0.97	Resistance
V-128xV17	-0.475**	-0.300**	1.56	-0.92	Resistance

** Significant at the level of 0.01

* Significant at the level of 0.05

Heterosis

Heterosis was calculated as the percent increase or decrease of F_1 performance above the mean performance of parental lines as well as over better parent for resistance for each cross. The magnitude of heterosis expressed as percentage increase or decrease over middle parent and better parent for character studied is presented in (Table 8). Heterotic response in the F_1 generation was observed for disease incidence on both original and transformed scales. On the original scale amongst resistant x resistant cross only V-335 x V-13 indicated heterosis both over mid parents and better parental values. The crosses involving V-13, V-17 and V-335 with susceptible lines indicated maximum heterosis for resistance. The negative sign of mid parents indicated that crosses were in the direction of resistance.

Gene action (Results based on original data)

Generally in the resistance crosses the effects were relatively larger than either additive or dominance effects. The mean effects were also larger than the absolute magnitude of epistatic effects (Table 9). While the m effects were highly significant for the eight resistant crosses the additive and dominance effects were significant for only 4 crosses of the a and d effects. The d effects were more important. The negative dominance gene effects for most of the crosses suggested that dominance was in the direction of resistance. In case of V-327 x V-13 followed by V-13 x V-128, the d effects was significant and had negative value.

Table 8: Mean values of parents and F1s and heterotic response for *Bipolaris maydis* (original scale)

		Mean Rating					Heterosis	
Crosses		P1	P2	MID. PAR	BET.PAR	F1	MID.PAR	OVER.BET.PAR(%)
CM-128xV-327	F1	2.92	3.48	3.20	2.92	3.08	-3.72	5.45
CM-128xV-335	F1	2.92	2.25	2.59	2.25	2.11	-18.29	-6.21
CM-128xV-13	F1	2.92	2.74	2.83	2.74	2.32	-18.02	-15.27
CM-128xV-128	F1	2.92	3.28	3.10	2.92	2.39	-23.06	-18.26
CM-128xV17	F1	2.92	2.10	2.51	2.10	1.87	-25.56	-11.15
V-327xV-335	F1	3.48	2.25	2.86	2.25	2.54	-11.43	12.59
V-327xV-13	F1	3.48	2.74	3.11	2.74	2.53	-18.48	-7.47
V-327xV-128	F1	3.48	3.28	3.38	3.28	3.11	-8.01	-5.33
V-327xV17	F1	3.48	2.10	2.79	2.10	1.83	-34.29	-12.90
V-335xV-13	F1	2.25	2.74	2.49	2.25	1.28	-48.63	-43.13
V-335xV-128	F1	2.25	3.28	2.77	2.25	2.04	-26.29	-9.44
V-335xV17	F1	2.25	2.10	2.18	2.10	1.41	-35.25	-32.97
V-13xV-128	F1	2.74	3.28	3.01	2.74	2.05	-31.80-	-24.98
V-13xV17	F1	2.74	2.10	2.42	2.10	1.51	-37.80	-28.25
V-128xV17	F1	3.28	2.10	2.69	2.10	1.56	-42.09	-25.87

Table 9: Estimate of gen actions effects for the 15 crosses for *Bipolaris maydis* (original scale)

Crosses		m	a	d	axa	axd	dxd
CM-128xV-327	F1	3.010ns	-0.340*	0.176ns	0.294ns	-0.124ns	-0.076ns
CM-128xV-335	F1	2.301**	0.687ns	-0.838*	-0.365*	0.707*	0.928*
CM-128xV-13	F1	2.584**	0.291*	-0.8988	-0.389*	0.399*	0.733*
CM-128xV-128	F1	2.842*	0137*	-1.077*	-.0.361ns	0.638*	0.330ns
CM-128xV17	F1	2.532**	0.506**	-1.860*	01.218*	0.197*	1.073*
V-327xV-335	F1	2.886*	0.154*	0.041ns	0.368*	-0.915*	-1.477*
V-327xV-13	F1	2.727**	0.493**	-1.057*	-0.483*	0.247*	1.333*
V-327xV-128	F1	3.555**	-0.026ns	-1.230*	-0.960*	-0.243*	0.677*
V-327xV17	F1	2.378**	0.880**	-1.270**	-0.314*	0.389*	0.364ns
V-335xV-13	F1	2.228**	-0.357**	-1.645*	-0.431*	-0.231*	-0.497*
V-335xV-128	F1	2.419**	-0.876*	-1.689ns	-0.962*	-0.721*	1.868*
V-335xV17	F1	1.833**	0.073*	-0338ns	0.430ns	-0.002ns	-1.011ns
V-13xV-128	F1	2.318**	-0.809*	-2.169**	-1.212*	-1.072*	3.277*
V-13xV17	F1	1.924**	0.050ns	-1.030*	-0.119*	-0.532*	0.407*
V-128xV17	F1	2.062**	0.420*	-0.907ns	0.227*	-0.338*	-0.191ns

m=F2a=additive axd=additive x dominance dxd= dominance x dominance
d= dominance axa= additive x additive
** Significant at 1% level
* Significant at 5% level

Each of the three digenic epistatic effects, in general, appeared to be important in the resistance crosses. Of these three effects, dd was playing a greater role followed by ad effects. The negative epistatic effects were in the direction of resistance. In V-335 x V-13 cross, dd effect were significant and negative. The negative value of dd effects indicated that were controlling the lesser manifestation of disease incidence. The relatively high magnitude and positive effects of dd in most of the crosses indicated that this interactions was enhancing susceptibility, while considering the CM-128 x V-335, CM-128 x V-13, CM –128 x V-128, CM-128 x V-17 and V-327 x V- 335 crosses were observed that ad and dd effects were highly significant and positive value. The positive sign associated with most of the estimates indicated that these epistatic effects were in the direction of susceptibility.

Results based on the transformed data

The interpretation of sign is reversed when the effects were obtained on the transformed scale. In V-335 x V-13 cross gave similar results to those obtained on the original scale. V-327 x V-17 and V-13 x V-17 crosses showed significant dominance gene effects and also negative dominance gene effects. Therefore, these crosses were in the direction of resistance. These results were also similar to those obtained on the original scale. In the crosses of CM-128 x V-335, CM-128 x V-13, CM-128 x V-128 and CM-128 x V-17. In CM-128 x V-335 the estimates of parameters m were highly significant while both a and d effects were significant in all crosses except for a effect. The ad and dd effects had positive value indicating dominance toward susceptible parent. Except for ad and dd effect in CM-128 x V-335 cross but these effects had positive value on the transformed scale. In case of the V-327 x V-17 the m effects were highly significant and both a and d parameters also were significant. The negative sign of d effects also indicated that the dominance effects were enhancing resistance. The estimates of parameter aa were more prominent. The negative sign of aa effects indicated that additive x additive effects were enhancing resistance. The results were same as obtained from original scale.

Maydis leaf blight caused by *Bipolaris maydis* is one of the destructive diseases of maize. The disease is prevalent in all warm and humid areas whatever maize is grown. Knowledge of the inheritance of disease is also must importance for the development of resistant genotype cultivars. Also for the adoption of an appropriate breeding procedure it is essential to understand the type of gene action involved. The present study was undertaken with an objective of understanding genetics of resistant of the disease.

The investigations were carried out both on the original (Ullstrup *et al.*, 1941) and transformed scale (Fisher and Yates, 1963). The results obtained on these two scales indicated only minor differences. Hence, in the discussion the results obtained on the transformed scale only will be taken into consideration. Analysis of variance revealed that the treatment effects were highly significant

for disease incidence. Highly significant between generation means were revealed for the characters under study. Highly significant differences were also observed within generation means. This indicated that the choice of parents was appropriate and that the parents were quite distinct in relation to the character studied (Table 4).

The concepts of combining ability were enunciated by Sprague and Tatum (1942). General combining ability was defined as the average performance of a line in several hybrid combinations and specific general ability as the deviation from the expectation based on average performance.

Griffing (1965) suggested that combining ability analysis could also be carried out by using further segregation generation. Combining ability computed on a diallel cross of six inbred lines of maize in the generations revealed that variances due to general and specific combining ability for *B. maydis* were highly significant. However, the variances for general combining ability were of a much higher magnitude. The GCA variance was higher in magnitude than SCA variance indicating the presence of additive effects for this trait but both GCA and SCA variance were significant the presence of dominance and over dominance was not ruled out.

Since general combining ability is a result of additive gene effects and SCA is dependent up on non-additive gene effects (Sprague and Tatum, 1942). It was concluded that additive gene action was playing the predominant role in this material. However, predominantly additive effects have been reported to contribute to the expression of reaction to this disease (Lim, 1975). Bogyo (1958) and Jha (1970) working with the inheritance of *H. turcicum* came to similar conclusions. Combining ability analysis is also useful in identifying parents with high general combining ability effects.

V-17 had the highest GCA estimate and was significantly different from other parents (Table 6). It also had distinctly lower disease reaction than other inbred lines and thus was the best line for the resistance to maydis leaf blight. It is suggested that this parent can be widely used in hybridization programme to achieve resistance. This parent when crosses also gave higher percentage of heterosis over better parent. V-335 and V-13 had the high and negative GCA estimate. These two parents also had distinctly lower disease reaction

The cross V-335 x V-13 will be of special significance as it can be exploited as commercial hybrid as well as for conventional breeding programme. V-128 x V-17 and V-327 x V-17 showed significant SCA effects. The three resistant lines, V-17, V-335 and V-13 had highest values for GCA effects, thereby indicating that these lines possessed the maximum combining for resistance in next generation. CM-128, CM-327 and V-128 had significant positive GCA effects. Thus they transmitted susceptibility to their progenies. Among them line CM-128 proved to be the poorest general combiner for imparting resistance. It was followed by V-327 and V-128.It is interesting to note that combinations between resistant lines, which were also good general combiners. It was reflected by relatively high and negative value (original scale)

for specific combining ability effects for single crosses between these lines. Most of the specific combining effects contributed by these combinations were not significant. High specific combining ability effects can be related with heterosis as in its expression both the dominance and the epistatic effects preponderate. If the crosses showing high specific combining ability involved both the parents, which are also good general combiners, they could be exploited for practical breeding. The present investigation revealed that highest estimated of SCA effects were observed in the crosses of V-335 with V-13 (resistant x resistant), which are good combiners, and they could be exploited for practical breeding.

Studies on gene action of quantitative traits should make use of different approaches to biometrical analysis, such as combing analysis (Griffing, 1956b) and estimates of gene effects (Hayman, 1958a). The combining ability is useful in identification of parents with high general combing ability effects and in detecting cross combinations showing high SCA effects. The estimation of gene effects made it possible to make a though study of the different gene effects in individual crosses, particularly in resistant x susceptible combinations. Estimation of genetics component from parental and F_1 data revealed that in the material under investigations both additive and non-additive gene action were important in the control of resistance. In the three resistant x resistant crosses the estimates of parameter m were larger in magnitude relative to parameter a and d or the absolute magnitude of epistatic effects. These results confirmed the findings from specific combining ability studies between resistant x resistant lines, where such combining did not impart resistant to the F_1 hybrid, superior to performance and was reflected in the relatively high and negative values for specific combining effects (Table 7).

The estimates of the six parameters for the various gene effects considered show that additive gene effects made the major contribution to variation in resistance to *B. maydis*. Epistatic effects were also important contributors to variation for resistance to *B. maydis* in the most of the crosses. The magnitude and significance of the estimates for aa, ad, and dd, over 15 crosses indicated that epistatic gene effects were present and important in the basic genetic mechanism of disease resistance in the population studied with regards to individual epistatic gene effects, additive x dominance and dominance x dominance effects appeared to contribute more to resistance to *B. maydis* in these crosses than do the additive x additive gene effects.

Dominance gene effects were exhibited by 5 of the 6 crosses. Its sign indicated the importance of dominance gene effects. The negative sign associated with these effects indicated that dominance was in the direction of resistance. The present investigation revealed that both additive and non-additive gene effects were involved in resistance to *B. maydis*. Reciprocal recurrent selection as suggested by Comstock *et al.,* (1949) would appear to be the best breeding approach in concentrating genes for resistance to *B. maydis* since epistasis, additive and dominance gene effects appeared to be important in the inheritance of resistance. There is hardly any literature available where studies either on

combining ability, estimation of genetic components or gene effects have been reported. Sharma *et al*. (1993) reported that dominance as well as epistasis both contributing to resistance along with additive gene effects, creates considerable hindrance in efficient utilization of additive gene in the enhancement of resistance level by any cyclic breeding procedures. Khehra *et al.* (1984) demonstrated the SCA component was found to be of greater importance then GCA component in the inheritance of maydis leaf spot. Inbred line 'H 3191' was best general combiner for disease resistance and also had high SCA variance. The nature of gene controlling resistance to *H. maydis* (race O) in some commonly used maize genetic stock. Some resistant genetic stocks, which have good combining ability and can be used in breeding programmes have also been identified (Saini *et al.,* 1979).

The exploitation of heterosis to raise the yield levels has been tried by several workers. The level of heterosis as well as selection advance in segregation generation to depend upon the genetic among the parents. The heterosis component is largely dependent on parental diversity as suggested by several workers in both self and cross pollinated crops. Therefore, the choice of diverse parents with good GCA is a pre requisite for carrying out an efficient hybridization programme. In practical heterosis, it is necessary to select combination with high degree of specific combining ability as well as parents with high general combining ability. In the present study it was shown that heterotic response in the F_1 generation was observed for disease incidence on both original and transformed scales. On the original scale amongst resistant x resistant cross only V-335 x V-13 indicated heterosis over mid parents and better parental values. The crosses involving V-13, V-17 and V-335 with susceptible lines indicates maximum heterosis for resistance. The negative sign of mid parents indicated that crosses were in the direction of resistance. Lim, (1975) reported that variation attributed to average heterosis, by line, and specific heterosis, between lines, were highly significant and resistance was partially dominant. Based on disease rating resistant inbreds contributed less heterotic effects for resistance to single crosses than did susceptible inbreds.

CONCLUSIONS

The analysis of variance revealed that the variance within parents and their progenies was highly significant for disease reaction studies indicating that the choice of the parents was appropriate for the present study. Analysis of variance for combining ability indicated that both GCA and SCA variances were highly significant for disease reaction studied. High GCA variance for disease reaction suggested operation of additive gene action. Out of the 6 parents 3 exhibited –ve and 3 exhibited +ve GCA effects for resistance to *B. maydis*. Resistance in inbred V-17 had the highest negative GCA effects hence it is the best general combiner for resistance.The estimates of SCA effects indicated several crosses showing -ve SCA effects. Out of the 15 hybrids only 5 hybrids showed significantly –ve SCA effects. Highest estimates of SCA effects were

observed in the cross V-335 x V-13 followed by V-327 x V-17 and V-128 x V-17. Parent V-13 (with significant negative GCA effect) when crossed with parent V-335 (with significant negative GCA effects) gave hybrid V-335 x V-13 which had significant negative SCA effects. Parent V-17 (with significant negative GCA effect) when crossed with parent V-128 (with significant positive GCA effects) resulted in a hybrid which had significant negative SCA effects. Amongst resistant x resistant cross only V-335 x V-13 indicated heterosis both over mid parents and better parental values. The crosses involving V-13, V-17 and V-335 with susceptible lines indicated maximum heterosis for resistance.

REFERENCES

Balmer E, Pereira O A P.1987. Doenças do milho. In: Paterniani E, Viegas G P. (Ed.) *Melhoramento e produção de milho*. 2.ed. Campinas: Fundação Cargill, v.2, cap.14, p.595-634.

Beck D L, Vasal S K, Crossa J.1990.Heterosis and combining ability of CIMMYT'S tropical early and intermediate maturity maize (*Zea mays* L.) germplasm. *Maydica*, v.35, p.279-285.

Bogyo T P, de Mueleneare H J H.1958.Preliminary investigation on the effect of the gene Y on the carotene content of maize seeding leaves.Proc.I Cong. *South Africa Genet.Soc*.77-79

Callaway M B, Smith M E, Coffman W R.1990. Diallel analysis of resistance to anthracnose stalk rot in maize inbreds. *Crop Science***,** v.30, p.335-337.

Carson M L, Stuber C W, Senior M L.1996. Identification of quantitative trait loci (QTLs) for resistance to two foliar diseases in a mapping population of recombinant inbred (RI) lines of maize. *Phytopathology*, v.86, p.59.

Carson M L.2001. Inheritance of resistance to *Phaeosphaeria* leaf spot of maize. Plant Disease**,** v.85, p.798-800.

Comstock R E, Robinson H F, Harvey P H.1949.A breeding procedure designed to make maximum use of both general and specific combining ability. *Agron. J*. 41: 360-367.

Crossa J, Vasal S K, Beck D L.1990.Combining ability estimates of CIMMYT's tropical late yellow maize germplasm. *Maydica*, v.35, p.273-278.

Dey S K, Mahajan V, Pal S S, Dhillon B S, Malhi N S, Khehra A S. 1989.Resistance to *Drechslera maydis* in maize inbred lines and their combining ability. *Indian Phytopath.* 42: 261-264.

Faluyi J O, Olorode O. 1984.Inheritance of resistance to *Helminthosporium maydis* blight in maize (*Zea mays* L.). *Theorical Apply Genet.* 67: 341-344.

Fisher R A, Yates F. 1963.Statistical Table for biological Agriculture and Medical Research. Oliver and Boynd Ltd.,Edinberg.

Griffing B.1956a.A generalized treatment of use of diallel crosses in quantitative inheritance. *Heredity*, 10: 31-50.

Griffing B.1956b. Concepts of general and specific combining ability in relation to diallel systems. *Aust. J.Biol.Sci*. 9**:** 463-493.

Griffing B.1958. Application of sampling variable in the identification of methods which yield unbiased estimates of genotypic variation components. *Aust. J. Biol*. 2: 219-245.

Hallauer A R, Miranda Filho J B.1988. Quantitative genetics in maize breeding. Ames: Iowa State University Press. 375p.

Han G C, Vasal S K, Beck D L, Elias E.1991.Combining ability of inbred lines derived from CIMMYT maize (*Zea mays* L) germplasm. *Maydica*, v.36, p.57-64.

Hayes H K, Immer F R, Smith D C.1955.Methods of Plant breeding. McGraw-hill book company, Inc.New York.

Hayman B I.1954*a*.A mathematical theory of gene action and interaction. Ph.D. Thesis, University of Birmingham Library.

Hayman B I.1958.The separation of epistatic from additive and dominance variation in generation of means. *Heredity*, **12**: 371-390.

Jha T D, Dhawan N L.1970.Genetic analysis of resistance to *Helminthosporium turcicum* Pass. in inbred lines of maize. pp.388-396 in S.P.Raychaundhuri *et al*.(Ed.)Plant disease problems. *Indian Phytopath. Soc*.,I.A.R.I., New Delhi.

Khehra A S, Rao, A V B S, Dey S K, Dehillon B S, Malhi N S.1984. Inheritance of resistance to maydis leaf blight in maize. *Indian J. agric Sci*. 54: 881-883.

Lim S M, Hooker A L.1976.Estimates of combining ability for resistance to *Helminthosporium maydis* race 0 in a maize population. *Maydica*, 21: 121-128.

Lim S M.1975.Heterotic effects of resistance in maize to *Helminthosporium maydis* race O. *Phytopathol.* 65: 1117-1120.

Lingam S S, Balasubramanian K A, Reddy P R R.1989.Combining ability analysis for resistance to *Helminthosporium turcicum* leaf blight in maize (*Zea mays* L.). *Annals of Agricultural Research*. 10: 405-409.

Nass L L, Lima M, Vencovsky R, Gallo P B.2000.Combining ability of maize inbred lines evaluated in three environments in Brazil. *Scientia Agricola*, v.57, p.129-134.

Pate J B, Harvey P H.1954. Studies on the inheritance of resistance in corn to *Helminthosporium maydis* leaf spot. *Agron. J*. 46: 442.

Payak M M, Sharma R C.1983. Disease rating scale in maze in India.Techniques of scoring for resistance to important disease of maize.All India co-ordinate maize improvement project. IARI. New Delhi, 1-4 pp.

Payak M M, Sharma R C.1985.Maize disease and approaches to their management in India.*Tropical Pest Management*. 31: 302-310.

Saini R G, Gurmukh Singh J, Grewal R k, Goal R K, Gupta A K.1979.Inheritance of resistance to *Helminthosporium maydis*(Race O) in corn.*Genet.Agr*.33:201-208

Sharma R C, Payak M M, Mukerjee B K.1993.Durable resistance in maize-A case study from India .In Durability of disease resistance.(eds .Jacobs,T. and Parlevliet,J.E).227-229 pp.Kluwer Academic Publishers,Dordrecht,Netherlands

Sharma R C.1983. Maydis leaf blight of maize. Techniques of screening for resistance to important disease of maize improvement project, IARI , New Delhi,5-15-pp

Silva H P.2001. Análise genética da resistência a *Puccinia polysora* Underw em milho (*Zea mays* L). *Piracicaba*: USP/ESALQ, 87p. (Tese - Doutorado)

Sprague G F, Tatum L A.1942.General v/s specific combining ability in single crosses of corn. *J.Amer.Soc.Agron*. 34: 923-932.

Thompson D L, Bergquist R R.1984. Inheritance of mature plant resistance to *Helminthosporium maydis* race O in maize. *Crop Science*, 24: 807-811.

Ullstrup A J.1941. Inheritance of susceptibility to infection by *Helminthosporium maydis* race I in maize. *J. Agric. Res.* 63: 331-334.

Van Eijnatten C L M.1961.Susceptibility of leaf blight caused by *Cochliobolus heterostrophus* in Nigerian varieties of maize. *Nature* 191: 515-516.

Vasal S K, Srinivasan G, Han G C, Gonzales C F.1992.Heterotic patterns of eighty-eight white subtropical CIMMYT maize lines.*Maydica*, v.37, p.319-327.